

Case report

Radiological description about the globally first case of human infected avian influenza virus (H10N8) induced pneumonia

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Abstract

Human infected avian influenza (H10N8) is an acute infectious respiratory tract infection caused by JX346-H10N8. The reported case in this paper is the globally first case report about radiological description of human infected avian influenza (H10N8) virus related pneumonia. The patient showed an epidemiological history of contacts to living poultries and the incubation period lasted for 4 days. The condition was clinically characterized by fever, cough, chest distress and obvious hypoxia. CT scan demonstrated the lungs with large flake of hyper-intense consolidation, confined patch of ground glass opacity, dilated bronchi, predominantly dorsal thickening of the interlobular septum, and other types of lesions related to interstitial pulmonary edema. Meanwhile, accompanying interlobar effusion, intrapulmonary effusion and pleural effusion were demonstrated in a small quantity by CT scan. Human infected avian influenza (H10N8) related pneumonia should be differentiated from pneumonia induced by human infected avian influenza viruses H5N1 and H7N9. No characteristic key points for radiological differentiation have been found. And its definitive diagnosis should be based on the etiological examination.

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Keywords: Pneumonia; Severe; Virus; Avian influenza A (H10N8); Radiology

1. Case introduction

A 73-year-old woman complained of cough with expectoration after catching a cold on Nov. 27, 2013. She experienced chest distress and shortness of breath after physical activities. Clinically, she showed no fever, no gastric pain and diarrhea, no urination frequency and urgency. At night on Nov. 29, 2013, she began to show fever and aversion to cold and was hospitalized on Nov. 30, 2013. The patient reported a medical

history of high blood pressure and coronary heart disease. In Dec. 2012, she was diagnosed with thymoma and received surgical removal of the neoplasm. Postoperatively, oral intake of pyridostigmine bromide was prescribed at a dosage of 60 mg for 3 times a day, which had favorable effects for symptoms control.

By physical examination on hospitalization, the patient showed a body temperature of 39.1 °C, respiration rate of 22 per min, and a blood pressure of 130/70 mmHg (1 mmHg = 0.133 KPa). She was conscious, with shortness of breath but no cyanosis. By auscultation, the both lungs showed coarse breathing sound but no wheezing, with fine moist rales on the right lung. The heart rate was 98 per min, with sinus rhythm. The abdomen was negative and the muscular strengths of the four limbs were normal.

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2. Clinical examinations and treatments

2.1. Epidemiological study

The patient reported a history of buying a silkie with her servant at a market on Nov. 23, 2013. The silkie was chosen by the seller, followed by processings such as plucking the feather at the stall. The patient stayed at the stall for about 5 min and left. After returning home, the servant was responsible for cooking the silkie. During the whole process of processing and cooking, the patient did not enter into the kitchen. The utensils for processing the uncooked silkie were immediately cleaned, including the knife and the chopping block. And they did well in separately using knives for cooked and uncooked food. After the surgery for thymoma, the patient never ate chicken and never drank chicken soup. The chicken soup cooked that day was drunk by the son, the daughter-in-law and the granddaughter of the patient. During 2 recent weeks before the onset of symptoms, the patient did not travel to other places and had no contacts to patients with fever and symptoms resembling to influenza. No other people paid their home visit. And the family members showed no symptoms resembling to influenza before the onset of the patient.

According to investigations, a total of 17 persons were confirmed with close contact to the patient. By medical observation, they showed no symptoms of fever and other respiratory symptoms. By throat swab test, the universal primers test of influenza A showed negative.

2.2. Laboratory test

On Nov. 30, 2013, the patient received laboratory tests, showing peripheral WBC count of $10.34 \times 10^9/L$, lymphatic ratio of 0.070, neutrophils ratio of 0.764, PLT count of $124 \times 10^9/L$, C-reaction protein (CRP) of $>200 \text{ mg/L}$. By blood-gas analysis, pH was 7.48, P_{O_2} 80 mmHg, and PCO_2 32 mmHg.

On Dec. 1, 2013, further laboratory tests showed Creatine Kinase (CK) 10 KU/L, myohemoglobin 62 $\mu\text{g/L}$, lactate kinase (LDH) 187 U/L. The renal function was examined with findings of Creatinine 35.7 $\mu\text{g/L}$. The test for hepatic function on Nov. 4 revealed ALT 25 U/L, AST 57 U/L, and ChE 1978 U/L. In addition, electrolytes were found to be K 4.2 mmol/L, NA 120.2 mmol/L, and Cl 88.8 mmol/L.

On Dec. 5, 2013, the Central Laboratory of Influenza in China confirmed that the patient showed the virus nucleic acid of human infected avian influenza (H10N8) positive. And the virus was nominated as A/Jiangxi-Donghu/346/2013 (H10N8), which was abbreviated as JX346. By sequencing and contrast, the virus has been demonstrated to be an influenza virus originally derived from poultry, with its internal gene from avian influenza virus (H9N2).

2.3. Radiological manifestation

On Dec. 1, 2013, chest CT scanning was performed, with findings of large flake of high-density consolidation opacity at

the right lung and patch of ground glass opacity at the left lung. In addition, the both lungs were demonstrated with thickening of interlobular septum as well as interlobar effusion in a small quantity. Pleural effusion was also revealed in a small quantity. Bed-side chest X-rays on Dec. 3, 4, 5, 2013 revealed that the lesions were increasing.

2.4. Clinical diagnosis and treatment

On Nov. 27, 2013, the patient experienced cough with whitish sputum after catching a cold, chest distress after physical activities, but no fever. On Nov. 30, 2013, she was hospitalized due to fever with a body temperature of 38.6°C . Laboratory tests on admission showed that the peripheral WBC count was $10.34 \times 10^9/L$, with lymphocyte ratio of 0.070, neutrophils ratio of 0.764, PLT count of $124 \times 10^9/L$, CRP of $>200 \text{ mg/L}$. The blood-gas analysis showed P_{O_2} $70 \pm 10 \text{ mmHg}$ and PCO_2 $20 \pm 10 \text{ mmHg}$. Medications of cefotaxime sodium and Levofloxacin were administered for infection and oxygen aspiration was prescribed. On Dec. 1, 2013, chest CT scanning was performed, revealing changes of severe pneumonia that included a large flake of consolidation opacity at the right lung and ground glass opacity at the left lower lung. After treatment, the patient still showed fever, decreased P_{O_2} , but increased PCO_2 . On Dec 2, 2013, tracheal cannulation was performed for respirator assisted ventilation, with aspiration of pinkish water like non-foaming sputum in a large quantity. The fever still persisted, with a higher body temperature and decreased P_{O_2} . On Dec 3, 2013, the virus nucleic acid test of influenza virus confirmed the diagnosis of human infected avian influenza virus (H10N8) infection and severe pneumonia. The previous anti-infection therapy was then continued together with anti-viral therapy of Tamiflu (Fig. 2). However, the high fever still persisted, with further decreased blood oxygen saturation. The following bed-side chest X-ray showed an increase of the lesions, indicating the condition was obviously progressing. On Dec 6, 2013, death occurred due to respiratory failure and shock.

3. Discussion

Pneumonia induced by human infected avian influenza virus (H10N8) is an acute infectious respiratory disease caused by a subtype of human infected avian influenza virus, JX346-H10N8 [1]. Its incubation period is about 4 days, which is similar to the incubation period of human infected avian influenza induced by H5N1 and H7N9. The disease is clinically characterized by fever, cough, chest distress and obvious hypoxia. On Dec 2, 2013, tracheal cannulation was performed for respirator assisted ventilation, with aspirations of pinkish water like non-foaming sputum in a large quantity, indicating the occurrence of pulmonary edema and uncorrected hypoxia. On Dec 3, 2013 (d 6 after onset), the virus nucleic acid test of avian influenza showed H10N8 positive, followed by administration of Tamiflu (Fig. 2). However, the symptoms were not relieved, with high fever and further decreased blood oxygen saturation. Bed-side chest X-ray also

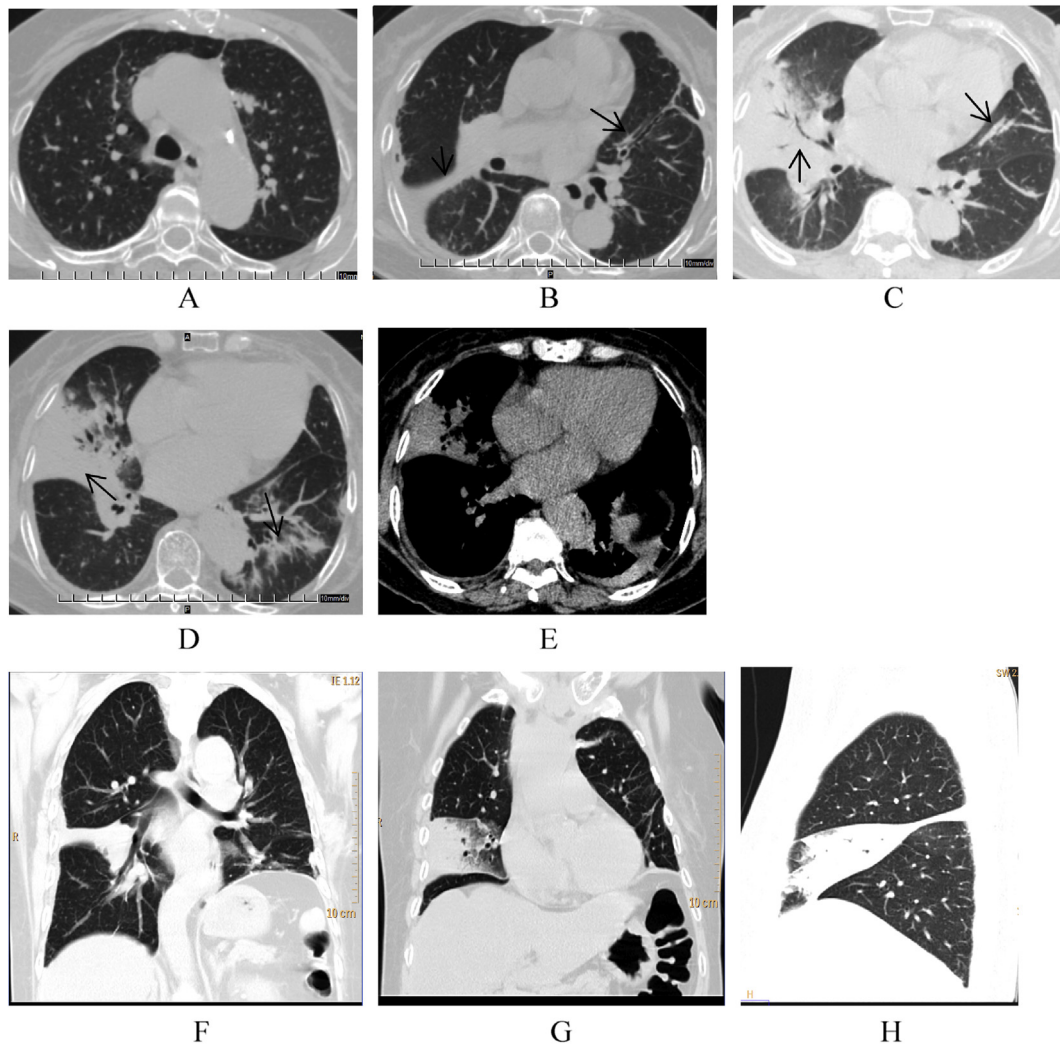


Fig. 1. (A–G). Chest CT scanning on Dec 1, 2013 (the 4th day after onset). A: A patch of high-density opacity at the left upper lung lobe near the aortic arch. B: The right interlobar effusion and pleural effusion in a small quantity, dilated bronchi at the left upper lung, and thickened interlobular septum (indicated by arrow). C: A large flake of consolidation opacity at the right middle lung lobe with air bronchogram, thickened interlobular septum at the left upper lung which was predominantly posterior to the interlobular fissure of the left lung (indicated by arrow). D: Consolidation opacity at the right middle lung lobe, patch of ground glass opacity at the dorsal segment of the left lower lung lobe. E: Intrapulmonary effusion of the left lung in a small quantity. F: Coronal image revealed obviously thickened right transverse fissure and right pleura. G: Coronal image showed consolidation opacity at the right middle lung lobe, which was peripherally obvious. H: Sagittal view image demonstrated co-existence of consolidation opacity at the right middle lung lobe and interlobar effusion.

revealed more lesions at both lungs, indicating the condition was progressing. On Dec 6, 2013 (d 9 after onset), the condition rapidly aggravated, with high fever, further decreased blood oxygen saturation, ARDS, septic shock, acute renal failure, and multiple organs dysfunction. Due to ineffective control of the virus infection, death occurred after emergency rescuing. The epidemiological data showed that the patient had a history of exposure to living poultry market. But the specimen for environmental surveillance showed no human infected avian influenza virus H10N8. The individuals with close contact to the patient received 2 weeks clinical observation, with no occurrence of influenza resembling symptoms. In addition, these individuals with a history of close contact to the patient showed negative to the virus nucleic acid test of H10N8, indicating that H10N8 cannot spread from person to person [2].

At d 4 after the onset, chest CT scanning revealed pulmonary consolidation opacity and pulmonary interstitial edema. The lesions are radiologically characterized by predominant distribution at both middle and lower lungs and mild condition at the both upper lungs (Fig. 1-A,C,D), large flake of high-density consolidation opacity by chest CT scan with obvious air bronchogram and confined patch of ground glass opacity (Fig. 1-C,D). In addition, chest CT scan demonstrated obvious bronchial dilation, thickening of the interlobular septum and other interstitial changes. The thickened interlobular septum is mainly distributed at the posterior and dorsal parts (Fig. 1-B,C), indicating interstitial pulmonary edema. Meanwhile, the patient showed interlobar, intrapulmonary and pleural effusions in a small quantity (Fig. 1-C,E). Coronal 3d reconstruction of the chest CT scans demonstrated obviously thickened transverse fissure of the right lung and obviously

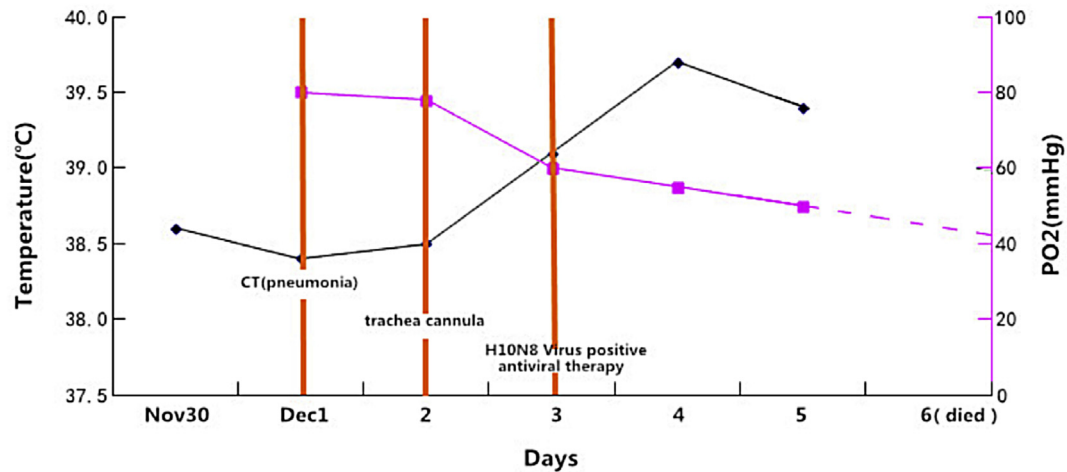


Fig. 2. Relationship between the body temperature and Po2 with tracheal incubation and anti-viral therapy.

thickened right pleura as well as obvious consolidation at the peripheral right middle lung lobe. Sagittal view image concurrently revealed consolidation of the right middle lung lobe and interlobar effusion. The lesions showed rapid progress. At d 6 after the onset, bed-side chest X-ray demonstrated the rapid progression of the lesions, with enlarged consolidation at the both middle and lower lung fields, large flake of consolidation at the right upper lung and left middle lung field, enlarged and blurry hilar opacity at both lungs. By tracheal cannulation, pinkish foaming sputum was aspirated. Clinically, the patient experienced hypoxia, which was in consistency with the radiological signs of pulmonary edema.

The disease should be radiologically differentiated from pneumonia induced by other subtypes of human infected avian influenza virus, such as H7N9 and H5N1 [3,4]. In terms of location, morphology and density of the lesions, the disease resembles to pneumonia induced by H7N9 and H5N1, with radiological manifestations of ground glass opacity and pulmonary consolidation, which are commonly observed at the both middle and lower lung fields. However, in the cases of pneumonia induced by H5N1, in addition to large flakes of ground glass opacity and consolidation at both lungs, the lesions distribute widely with rapid progression. Some lesions are even migratory, which is characteristic radiological sign of pneumonia induced by H5N1. And the lesions are absorbed slowly, with obvious interstitial fibrosis during 5 years follow-ups [3,5]. In the cases of pneumonia induced by H7N9, the lesions are predominantly distributed at both middle and lower lungs, which are commonly large flakes of ground glass opacity. Compared to pneumonia induced by H10N8, pneumonia induced by H7N9 showed milder interstitial changes at both lungs, with slower changes and absorptions but a high mortality rate of up to about 36% [4,6].

Generally speaking, pneumonia induced by human infected avian influenza virus H10N8 is radiologically characterized by

pulmonary consolidation, ground glass opacity, interstitial edema at both lungs and accompanying interlobar, intrapulmonary and pleural effusions in a small quantity. It should be differentiated from pneumonia induced by human infected avian influenza viruses H5N1 and H7N9. And the definitive diagnosis should be based on the epidemiological history and etiological examination.

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